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THE OCTOPUS MOTIVE IN ANCIENT CHIRIQUIAN ART

By George Grant MacCurdy
PEABODY MUSEUM, YALE UNIVERSITY
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The ancient art of the one small province of Chiriqui in the diminutive Republic of Panama is perhaps better known than that of any region of like size in the New World. The material for study is abundant, consisting largely of ceramic products, both painted and unpainted. These have become more or less familiar to students of archeology through two large monographs, one by Professor W. H. Holmes[1] and the other by myself.[2]

It was found that the pottery could be readily divided into about a dozen rather distinct groups, depending largely on the nature of the paste and other materials used as well as the method of producing the dominant decorative features. For example, symbolism and ornament in the unpainted ware find expression in plastic forms and incised patterns. Both are traceable to zoömorphic originals, as are the plastic and painted motives in the painted ware. The motive may represent the entire animal in fairly realistic fashion or it may consist of almost any part of the animal, as for example the head, foot, tail, eye, appendage, or some characteristic body marking. Certain animal motives are always represented in the round or in relief; others appear only as incised patterns and still others predominantly as painted forms.

Thus we find the plastic armadillo dominating the great group that might appropriately bear that name; the incised serpent goes with a distinctive group of black ware; the plastic fish in the guise of tripod supports runs through another group; while the painted alligator is supreme in two closely related groups of painted ware.

One soon learns by experience to associate a given motive with a given paste, slip, quality of modeling, and the character and number of the

colors employed, as well as the method of their application. After the armadillo ware, perhaps the largest group is the one to which Holmes gave the name *lost color* ware, the designs being produced by the removal of color rather than by its direct application. In addition to the process of negative painting employed, this group is also characterized by distinctive forms as well as the nature of the paste and the colors, also the degree of finish (or absence of it) to which the modeling was carried.

In the lost color ware a majority of the designs consists of rhomboidal figures, triangles, associated bands composed of groups of straight lines, and designs in the shape of fronds and waving arms. They are found alone as well as in combination. The fronds, waving arms, triangles, and straight bars, as well as the lozenge-shaped designs are often associated with series of dots. What is the meaning of all this? At first glance they seem far removed from the motives derived from animal forms so characteristic of other groups of Chiriquian pottery. Could they be plant derivatives? Are they perhaps simply the products of uncontrolled fancy?

A key to the mystery recently came to light in the shape of a more realistic rendering of the motive than had been known hitherto. It consisted of a lozenge-shaped body to which was attached eight waving arms. It filled a circular panel on the two sides of a round-bodied lost color vase collected by Mr. George G. Heye while on a trip to Chiriqui in 1913. This vase to which Professor Marshall H. Saville had called my attention was recently published by me.[3] The design represents an octopus (fig. 1). At that time, I pointed out its kinship to many designs previously published by Holmes and myself, but the significance of which had not been understood.

A further study tends not only to confirm what was said in my last note but also to emphasize the importance of this newly discovered motive as a special feature of lost-color symbolism in ancient Chiriquian art, and as another example of how a whole group of related motives in primitive art may be traced to a single zoömorphic prototype.

Recently in going over some duplicate Chiriquian pottery with a view to making an exchange, I came across several new variations of the octopus motive. In one shapely vase the eight octopus appendages are evenly distributed over the upper zone, being attached to a line just below the neck of the vessel (fig. 2). The neck and aperture thus take the place of the octopus body and mouth. The artist's point of view can perhaps be better appreciated by looking down on the vase from above rather than by a side view.

A similar idea but expressed in a different way is shown in figure

3 (fig. 167 of Holmes's monograph). Here the arms are attached to the equatorial zone (or slightly lower) and rise like short-based triangles to the neck of the vase. They are dotted to represent the succers. In looking down upon this vase the arms converge toward the narrow neck of the vessel, which thus becomes the mouth opening of the octopus. The body of the vase therefore becomes the body of the octopus, which is represented not only in painted design but also in the round.

FIG. 1.—OCTOPUS MOTIVE AS A CIRCULAR PANEL DECORATION. HEYE COLLECTION. CAT. NO. $\frac{3}{8}\frac{8}{1}\frac{1}{1}$.

Thus the octopus appendage may be represented in two ways; as a frond-like arm or as a short-based triangle. Each type is often (but not always) accompanied by dots representing the succers. Both types are seen in figure 4. Four of one kind alternate with four of the other kind converging toward the neck of a small globular vase with black ground and cream pattern. The four fronds are straight, each consisting of two lines enclosing a row of dots. Each triangular arm is set in a series of paired converging lines the outer ones being accompanied by succer dots. An identical octopus design is repeated on the lower half of the vase, beginning midway and with the eight arms converging toward a central point on the bottom.

FIG. 2.—OCTOPUS MOTIVE, THE EIGHT APPENDAGES FORMING THE SHOULDER DECORATION, THE NECK OF THE VASE REPRESENTS THE OCTOPUS BODY. YALE COLLECTION. $\frac{1}{4}$.

Akin to all the foregoing is an octopus motive produced by shifting

the mouth opening from the neck of the vase to an equatorial point on the side of the body and representing it by means of a painted circle. In order to eliminate as it were the neck and aperture of the vase from consideration, a large circular panel is formed about this make-believe mouth opening as a center. The eight arms converging from the periphery of the panel toward the central mouth opening produce the same effect as though one were looking down on the vase in figure 3. This gives the design that in earlier publications was called the rosette. It is simply a variety of the octopus motive and is met with perhaps oftener than any other single variety. In order the more easily to arrive at a circular panel, the body of the vessel was made spherical and

FIG. 3.—OCTOPUS MOTIVE. THE SUCCERED ARMS RISE LIKE SHORT-BASED TRIANGLES TOWARD THE NECK OF THE VASE WHICH SERVES AS THE OCTOPUS BODY. FROM HOLMES.

FIG. 4.—TWO VARIETIES OF THE OCTOPUS APPENDAGE MOTIVE. FOUR ARMS OF EACH KIND RISE TOWARD THE NECK OF THE VASE; THIS DESIGN IS REPEATED ON THE LOWER HALF OF THE VASE. HEYE COLLECTION. CAT. NO 7461. ½.

the neck small, two characters common to lost color vases. It is therefore probable that the exigencies of the design tended to control the shape of the vessel, and vice versa. A good example just rescued from the duplicate material in the Yale Museum is reproduced in figure 5.

Referring back to the key specimen (see fig. 1), we find the octopus body represented by a rhomboidal or lozenge-shaped figure. In some realistic examples showing appendages attached to the body, the dots representing succers are placed within the field of the body rather than on the appendages. Remembering the freedom with which the ancient Chiriquian artist suppressed or transposed parts, one would expect to find cases where the body is represented and the appendages omitted. This would give an octopus body motive. The body motive, as was the case with appendage motives, is repeated to form zonal or other

ornaments (fig. 6). As might be expected, it is not limited to the lozenge form. Any four-sided, perhaps even rounded or triangular design would answer the symbolic requirements, especially if it contained dots to suggest succers and, by inference, the appendages on which they grow.

With such an exuberant proliferation of motives derived from a single zoömorphic original, there is of course ever present the possibility of the overlapping of motives that started from wholly different originals. I have already referred to the occurrence of the dorsal-view motive of the alligator on lost color ware (*Op. cit.*). It is highly probable that the overlapping of this motive (perhaps also the scale-group and spine

FIG. 5.—OCTOPUS MOTIVE. THE TWO KINDS OF APPENDAGES CONVERGE TOWARD THE CENTER OF A CIRCULAR PANEL. YALE COLLECTION. ½.

FIG. 6.—ZONAL DECORATION CONSISTING OF THE OCTOPUS BODY MOTIVE ENCLOSING SUCCER DOTS. YALE COLLECTION. ½

motive) and the one derived from the succers and appendages of the octopus has taken place to some extent, due to the convergence toward a common type, of scale-spine symbols of the alligator on the one hand and appendage-succer symbols of the octopus on the other. In so far as ancient Chiriquian art may serve as a guide, however, such overlappings instead of accounting for the evolution of the various motive groups, are rather to be considered as exceptions that prove the rule.

[1] Ancient art of the province of Chiriqui, *Smithsonian Inst., Rep. Bur. Amer. Eth.* 6 (1888).

[2] A study of Chiriquian antiquities, *Mem. Conn. Acad. Arts Sci.*, 3 (1911).

[3] Note on the archeology of Chiriqui, *Amer. Anthrop., N. S.*, 15, 661–667 (1913).

THE LIFE CYCLE OF TRYPANOSOMA BRUCEI IN THE RAT AND IN RAT PLASMA

By Rh. Erdmann

OSBORN ZOOLOGICAL LABORATORY, YALE UNIVERSITY

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It is well known that Trypanosoma brucei does not appear in the peripheral circulation until about forty-eight hours after inoculation. The actual history of the organism during this period has not yet been established for Trypanosoma brucei; and in other trypanosomes, in spite of numerous investigations, it has not been fully elucidated (cf. Minchin,[1] and Carini[2]). It has been impossible to study the uninterrupted life history of the trypanosome in the mammalian host.

The method of cultivating Trypanosoma lewisi *outside* the mammalian host was first discovered by Novy and McNeal in 1903.[3] McNeal[4] states that Trypanosoma brucei changes its form in the culture medium into round bodies which are not capable of causing infection in the rat. Laveran and Mesnil[5] show four stages of this transformation of trypanosomes into round bodies in a medium of serum and horse blood. These authors term these forms "mis en boule" and believe that they are capable of starting a new infection and that they are comparable, when submitted to unfavorable conditions, to the 'latent bodies' found by More and Breinl[6] in Trypanosoma gambiense and later in Trypanosoma equiperdum and Trypanosoma lewisi.[7] These mammalian trypanosomes showed, according to these authors, cyclical changes in the number of trypanosomes in the host and the periodic appearance of the 'latent bodies.' Thus there are periods in which many trypanosomes are in the peripheral *blood* and periods in which few or none can be discovered. These round forms have never been seen in the mammalian *blood* in Trypanosoma brucei, but only in the above mentioned culture media.

For the purposes of the present study, which demand a continued control of the trypanosomes in culture which is not possible by cultivation in test tubes, I have cultivated Trypanosoma brucei in rat plasma on slides, using the tissue-culture methods of Harrison, Burrows, and Walton.[8] The plasma was either inoculated with the trypanosomes themselves or used as a medium for the growth in vitro of various infected tissues of the host. In the plasma of the rat I have been able to keep the trypanosomes in a normal condition for an indefinite period, whereas by the use of Ringer's fluid or blood bouillon the organisms die after a few days.[9]

For one strain of trypanosomes, herein designated as 'M,' with which the experiments were made I am indebted to Prof. F. G. Novy of the University of Michigan. It is the same Nagana material which was used by Kanthack, Durham and Blandford, and Laveran and Mesnil for so many years. A second strain, 'R', was secured from Dr. W. Brown of the Rockefeller Institute. However, since both strains showed the same changes in the plasma medium, I shall confine myself chiefly to a description of strain M. Also, in describing the morphology of the forms studied, I shall refer, in this paper, to the larger nucleus merely as 'nucleus,' and the smaller nucleus as blepharoplast, thus avoiding any discussion as to the fundamental origin or theoretical significance of these structures.

It will be necessary to keep in mind some general observations concerning the growth of this well known strain (M) *in the rat*, in order to interpret some of the phenomena observed in the plasma. The period in which the strain kills the rat after infection is generally three or four days in length, when 2 cc. of Ringer's solution, with six drops of blood taken from an animal shortly before its death, is used. When *one* trypanosome is used for inoculation, the lethal period lasts five days.[10] At the end of this period, shortly before death, there appear generally only those forms which have a well marked nucleus and are known as 'Schmalformen.' Schilling[11] mentions them at this period, and Oehler[12] considers them as 'Wucherformen.' Observed with dark-field illumination, 72 hours after infection, these animals appear as shown in figure 1, f and g. Text figure 1, d and e, represents trypanosomes 62 hours after inoculation. These animals are larger because they are probably not in such a rapid period of multiplication as animals f and g. Forty-eight hours after inoculation animals of the type figured in figure 1, b and c, can be discovered. These forms are the so-called 'Breitformen,' which are distinguished by their wealth of granules. Since Kühn and von Schuckmann[13] rarely observed forms filled with granules it would seem that they must have taken the material for their excellent cytological studies shortly before the death of the infected animal.[13] In strain M, after 48 hours infection, broad forms with granules prevailed, but forms without granules could also be detected. Trypanosomes which are about to die often show a granular appearance, but those observed in plasma, and figured in figure 1, a, b and c, were living and moving in the manner characteristic of Trypanosoma brucei. Thirty-six hours after subcutaneous inoculation the trypanosomes in the peripheral blood are very scarce. An example of an animal from the blood at this period is shown in text figure 1 a. The characteristic shape of the trypano-

Fig. 1

Fig. 3

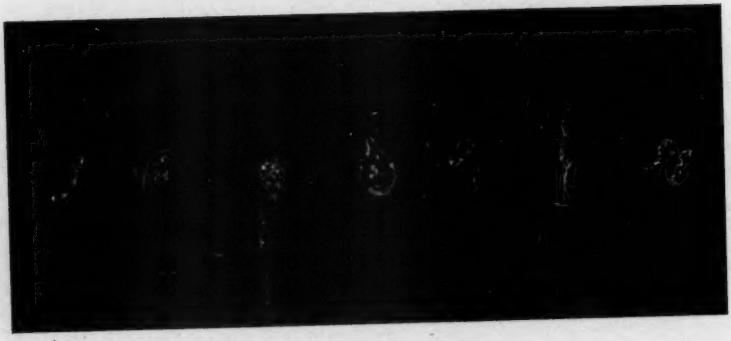

Fig. 6

somes is not yet assumed and the nucleus is not yet distinguishable. There remains, therefore, an interval of 36 hours after the new inoculation of strain M in which no typical trypanosomes could be discovered in the peripheral circulation. Crawley[14] shows that long continued

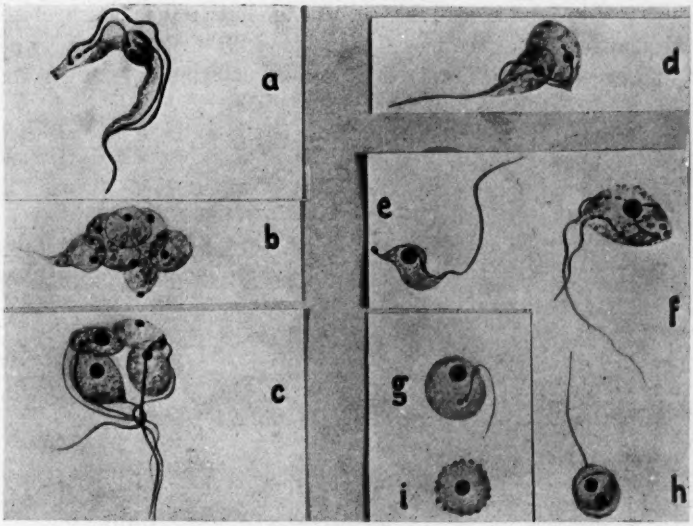

Fig. 2

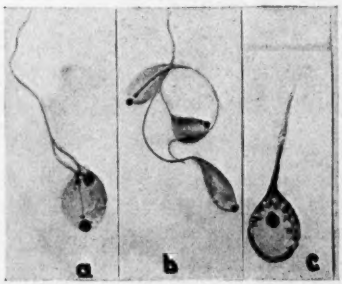

Fig. 4

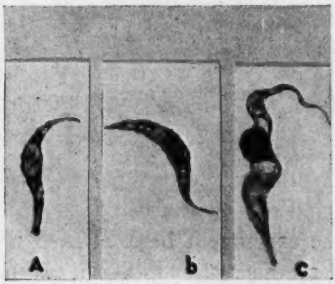

Fig. 5

use of dark-field illumination is inimical to protoplasm. Consequently great care must be taken in interpreting the different elements of the blood. The use of plasma instead of blood obviously eliminates many difficulties, because only a few elements of the blood are transferred into this culture medium.

DESCRIPTION OF EXPERIMENTS

The following short description of three typical experiments from a series of thirteen which were performed from September 25, 1914 to April 27, 1915, indicates that Trypanosoma brucei in slide plasma cultures undergoes different changes when maintained at a temperature identical with that of the mammalian blood (37°C.) from those which they undergo when maintained at a temperature (16°–18°C.) comparable with that of an invertebrate host.

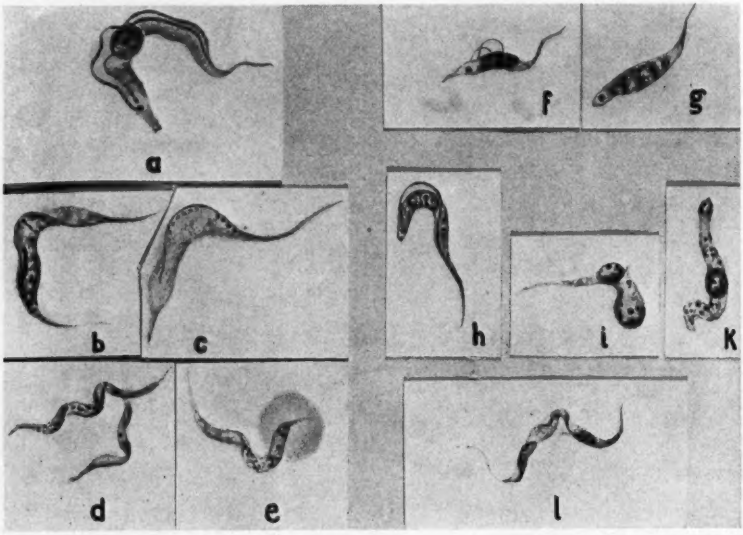

Fig. 7

Strain M, October 12 to 19, 1914. Culture kept at 37°C.—For this experiment the trypanosomes used resembled those of figure 1, animals f and g. The rat had been inoculated 84 hours, and died 2 hours after the experiment began. In figure 2, a trypanosome is pictured which has been six hours in the plasma medium. It was stained, after fixation with Schaudinn's fluid, with Giemsa feucht. This animal has the typical aspect of a trypanosome and proves that trypanosomes can live and divide in plasma at least six hours without changing their typical forms. Incidentally it has been observed in dividing forms just mentioned that the *nuclear* division lasts 2½ hours.

After having been subjected to the plasma for 24 hours the trypanosomes divide more rapidly. One organism frequently produces four

or eight. Figure 2 b shows such a multiple division. In these specimens only nuclei are present, but several hours later almost every animal has a nucleus and a blepharoplast. The latter individuals grow and divide (fig. 2 e). The nucleus has become smaller, the blepharoplast is still lying in the posterior end of the animal, but the distance between them has been diminished. Also the undulating membrane has disappeared. Division into two animals is most frequent on the third and fourth day (fig. 2 d and f), and generally on the fifth day small individuals (fig. 2 e) with somewhat trypanoform appearance are numerous. On the fifth day round forms appear (fig. 2 g). That they are capable of division is shown in figure 2 h. These forms on the fifth and sixth day become motionless, and the flagellum encircles the animal. Often the staining capacity of the flagellum partially disappears, as is shown in figure 2 i. It is not necessary that the formation of these 'involution stages' last six days since sometimes three days after the trypanosome is taken from the rat, the stage as figured in figure 2 e is attained.

These small forms are, by *dark-field illumination*, actively motile, and finally after the flagellum encircles the cell, gradually come to rest. The nucleus appears yellowish, the blepharoplast has a reddish yellow tinge. These characteristic stages are the last forms visible in plasma culture at 37°C.

It seems probable that these forms found at the end of the cycle in the plasma similarly occur in the *mammalian* host, and in developing there give rise to the recurrent cycles of trypanosome form. This would be a general confirmation of the views of Moore and Breinl, that 'latent bodies' observed by them in blood are the cause of the cyclical appearance of those trypanosome forms which they studied, i.e., Trypanosoma gambiense, Trypanosoma equiperdum, and Trypanosoma lewisi. This view affords plausible explanation of the reappearance of trypanosomes in the blood, and would eliminate the necessity of accepting a theory which implies the occurrence of parthenogenesis.[15]

Strain M, February 13 to March 13, 1915. Culture kept at 16° to 18°C.—After being 62 hours in the rat, the trypanosomes were transferred to the plasma medium, and kept at a temperature of 16° to 18°C. At this temperature the changes in the plasma medium do not proceed in as rapid a manner as at 37°C, the temperature of the mammalian blood. These were chiefly forms *without* granules (fig. 1, d and e). Figure 3, a and b, represents animals which have been forty-eight hours in the *plasma*. In these forty-eight hours no form changes are revealed by dark-field illumination, but frequently forms without a discernible nucleus are present (fig. 3 b). The wealth of granules is remarkable,

and divisions were noticed. The following day small actively motile forms appear (fig. 3, c and d). These are all without discernible nucleus when observed by dark-field illumination. In subsequent days these forms become smaller and do not change for an unlimited period, provided the organisms are transferred into new plasma under sterile conditions. Often they seem motionless, then again they have a corkscrew-like motion in the plasma. Frequently they flatten out, like a disk, or their edges fold over.

The preparations of preserved specimens show divisions into four and eight individuals as well as single individuals and, very rarely, the round forms, such as have been already described from the higher temperature (figure 2, b, c, d, e, f, g and h). Besides these forms similar to those described in plasma at 37°C., *other* types were found at 16° to 18°C. (fig. 4, a, b and c). Pear-shaped individuals are observed lying in clusters of from four to eight. The relative position of nucleus and blepharoplast has become changed in some of the animals observed at this period. The basal granule could not be distinguished, but the flagellum seems to arise from the blepharoplast. Figure 4, a shows such an animal, which perhaps might be termed crithidia-like because of the nucleus and the blepharoplast and the absence of an undulating membrane. This is the most frequent form in the plasma, and may be found even 30 days after the plasma is inoculated. These forms divide but undergo no further morphological changes.

Rounded forms of the above mentioned type (fig. 2, g and h), and of a slightly different type can also be observed on the fifth, sixth and seventh day after inoculation into the plasma (fig. 4, c). In these the flagellum has disappeared and only a tail of protoplasm is to be seen. They have a slight resemblance to the Dauerstadien in the frog trypanosome described by Doflein[15] except that in the latter forms the protoplasmic 'tail' has disappeared. In brief then, the full grown trypanosomes, in the plasma, at a temperature of 16° to 18°C., undergo changes which frequently lead to the formation of crithidia-like forms, and also sometimes, but rarely, to rounded forms.

After discovering that the crithidia-like and the rounded forms do not undergo further changes in the plasma (they were in the plasma from February 13 to 21), I inoculated them again into the rat. Five days after this inoculation small forms were observed (fig. 5, a, b). These were full grown at the seventh day (fig. 5, c) and caused the death of the rat at the ninth day of infection. Figure 6, which presents animals from another but exactly similar experiment, gives a good idea of these forms, which are present in the rat's blood on the fifth, sixth and

seventh days after the crithidia-like forms are reinoculated into the rat. The individuals shown in figure 6, f and g, are probably identical with the forms illustrated in figure 5, a and b. It is evident from these figures that animals with typical trypanosome form are not present in the blood at this period. However, that the forms which are present develop in the blood into Trypanosoma brucei has been proved by four experiments in which the inoculation of crithidia-like forms was carried out (fig. 5 c). The typical trypanosome forms did not always appear after the first inoculation of infected plasma into the rat. Sometimes two or three passages were necessary to effect this phenomenon—but sooner or later Trypanosoma brucei with its characteristic form was present. Every precaution was taken to exclude possible sources of error by contamination, etc. The blood of all rats employed was examined before inoculation for Trypanosoma brucei. The animals were kept in a room where animals with Trypanosoma brucei had never been before, and further, the rats were etherized to eliminate the highly improbable source of infection, the rat flea. This new strain M proved highly virulent and caused the death of the rat in three days.

An attempt was made to determine in what organ of the rat the crithidia-like forms and the rounded forms underwent transformation into the typical trypanosome form. Pieces of spleen, liver, heart and lungs of rats, infected with these forms, were implanted into plasma and kept alive and growing for a considerable time. Study of these tissue cultures failed to reveal any full grown trypanosomes. Only twice were even crithidia-like forms found in a culture of lung tissue. It is necessary for these experiments to be repeated on a larger scale for it is certain that rats infected with crithidia-like forms carry them, or young trypanosomes, for a considerable time. It is quite probable that this phenomenon goes on in nature and that some wild animals are the carriers of the infection by means of the form under discussion, even where full grown trypanosomes cannot be detected.

Finally, I shall briefly describe some changes in Trypanosoma brucei in plasma which I have succeeded in obtaining but once. The experiment was started in the same way and under the same conditions as outlined above, the plasma being kept at 16° to 18°C., etc. After twenty-four hours numerous trypanosomes without definite nucleus appeared (figure 7, b, c, d, e). They resemble the forms of Trypanosoma brucei which Bradford and Plimmer[17] observed in the lungs. These animals I found dividing rapidly until at the third day two types were present as shown in figure 7, f and g. The following day also two different types could be distinguished: animals with a clearly defined nucleus, but with

no trace of a blepharoplast or flagellum (fig. 7, i and k), and animals with nucleus, blepharoplast and flagellum (fig. 7, h). They were much smaller and narrower than the typical Trypanosoma brucei. The significance of this temporary disappearance of the nucleus is not clear. Such animals might be compared with von Prowazek's so-called 'indiferrente Formen.' However, the two different types which arise from these animals are not degeneration forms because they give rise to small trypanosomes (fig. 7, l) about eleven days after inoculation into the plasma. The appearance of the above mentioned types gives the only suggestion of dimorphism, which some might interpret as sexual in nature, which has appeared in my experiments.

Summary.—It is believed that the method employed in these experiments—which is a modification of tissue culture methods and excludes the possibility of the presence of confusing flagellate organisms—affords the means of following, outside the body of the invertebrate host, the sequence of changes in the life of trypanosomes.

Thus far its use has resulted in the discovery of dimorphic forms, latent or round forms, and crithidia-like forms in Trypanosoma brucei outside of the invertebrate host. The crithidia-like forms, when reinoculated into a rat, give rise to typical Trypanosoma brucei from which they had taken their origin.

[1] Minchin, Protozoa, London, 1912, p. 202; Minchin and Thomson, *Q. J. Microsc. Sci., London*, 60 (1915).

[2] Carini, *Ann. Inst. Pasteur, Paris*, 24, 149 (1910).

[3] Novy and McNeal, *Contrib. Med. Res.*, 1903; *J. Inf. Dis.*, 1 (1904).

[4] McNeal, *J. Inf. Dis.*, 1, 535 (1904).

[5] Laveran and Mesnil, Trypanosomes et Trypanosomiases, Paris, 1912, p. 459; *Ann. Inst. Pasteur*, 15 (1901).

[6] Moore and Breinl., *Ann. Trop. Med. Parasit.*, 1 (1907).

[7] Moore and Breinl, *Ann. Trop. Med. Parasit.*, 2 1908); *Proc. R. Soc.*, B. 80 (1908)..

[8] Harrison, *Proc. Soc. Exp. Biol. Med.*, 4 (1907); *J. Exp. Zool.*, 9 (1910). Burrows, *J. Amer. Med. Ass.*, 55 (1910); *J. Exp. Zool.*, 10 (1911); Walton, *Proc. R. S.* B. 87, 452 (1914).

[9] Erdmann, *Proc. Soc. Exp. Biol. Med.*, 12, 57 (1914).

[10] v. Prowazek, *Centralbl. Bakt., Jena*, 68 (1913).

[11] Schilling, *Berlin, Arb. Gesundhtsamt.* 21, 188 (1904).

[12] Oehler, *Zs. Hyg., Leipzig*, 78, (1914); *Heidelberg Sits Ber. Ak. Wiss.*, 1911.

[13] Kühn und v. Schuckmann, *Zool. Jahrb., Jena, Suppl.*, 1912, p. 338.

[14] Crawley, *Bull.* 119, *Bur. An. Ind., U. S. Dept. Agric.*, 1909, p. 6.

[15] v. Prowazek, *Berlin, Arb. Gesundhtsamt*, 22 (1905).

[16] Doflein, *Arch. Protistenk.*, 19, 222 (1910); *Freiburg, i. B., Ber. nat. f. Ges.*, 20 (1913).

[17] Bradford and Plimmer, *Q. J. Microsc. Sci.*, 45 (1902). Pl. 21, figs. 6, 7, 8, 14.

THE EFFECT OF PRESSURE ON POLYMORPHIC TRANSITIONS OF SOLIDS

By P. W. Bridgman

JEFFERSON PHYSICAL LABORATORY, HARVARD UNIVERSITY

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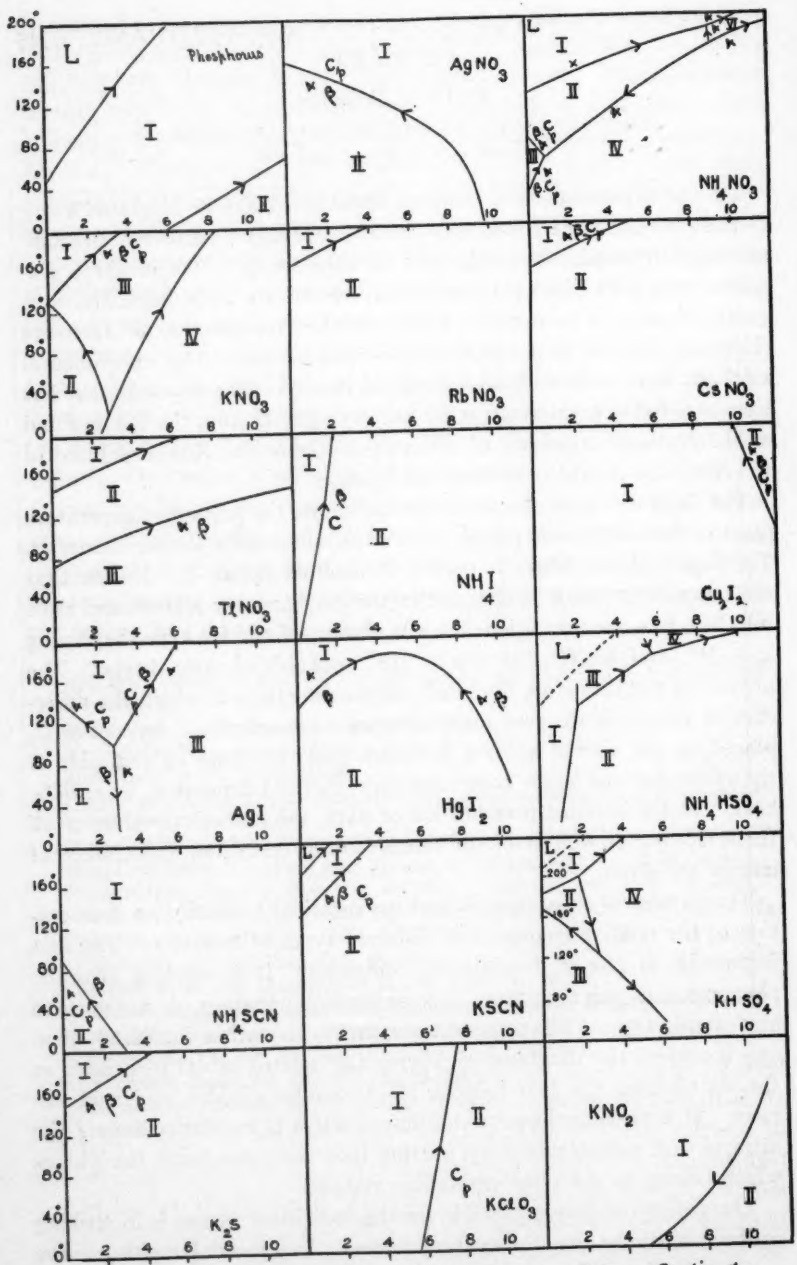
This note presents in a compact form by means of diagrams many of the essential facts concerning the effect of high hydrostatic pressure on the polymorphic transitions of 30 substances. Five of these diagrams have been previously published,[1] eleven are to be published with greater detail in a forthcoming number of the *Proceedings of the American Academy*, and the rest await detailed publication. The experimental methods have been fully described previously. The investigation has been assisted in great measure by generous grants from the Bache Fund of the National Academy of Sciences and from the Rumford Fund of the American Academy of Arts and Sciences.

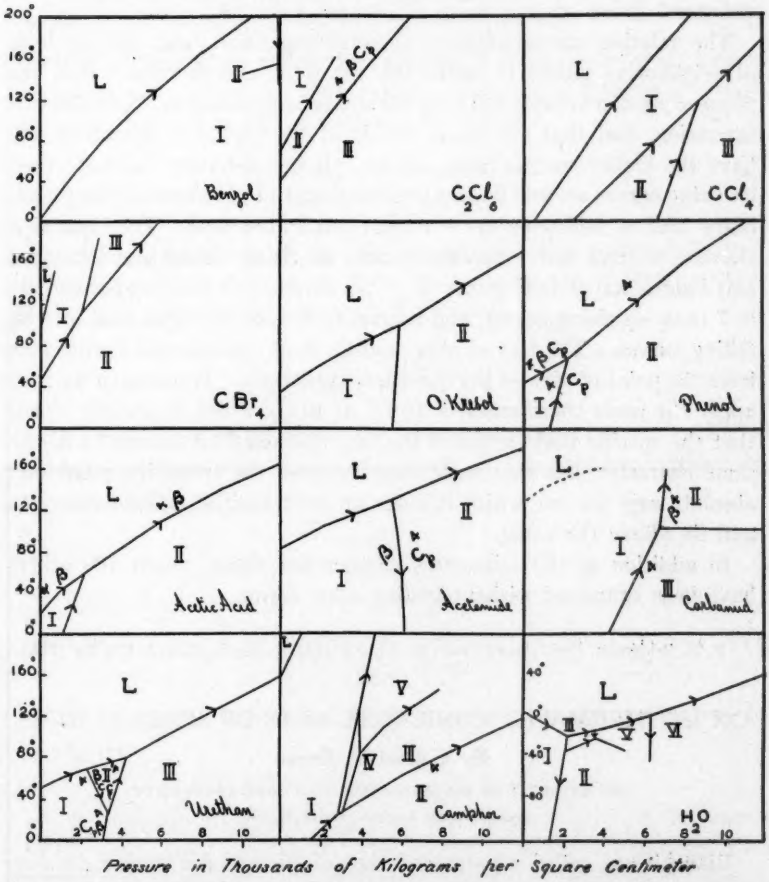
The diagrams show the transition curves on the pressure-temperature plane of the several solid phases, which are indicated by Roman numerals. The liquid phase, where it occurs, is denoted by an *L*. Notice that the temperature scale is changed in the diagrams for $KHSO_4$ and H_2O. NH_4NO_3 has one transition line not shown, of the ice type, beginning at $-16°$, and $RbNO_3$ has one at 219°, probably of normal type. The arrows on the transition lines indicate the directions in which the difference of volume of the two phases decreases numerically. An α, β or C_p placed on one side of a curve indicates that the phase on that side of the curve has the larger compressibility, thermal expansion, or specific heat. In the detailed presentation of data, the numerical values of all these factors, as well as of the latent heat of transition and change of energy are given.

The number of substances is perhaps sufficient to justify an enumeration of the relative frequency of different types of behavior. The first impression is one of bewildering complexity, it is obvious that the phenomena of polymorphism, even under high pressures, do not tend to any simple type. The apparent complexity is rather increased when one considers the diagrams of chemically related substances, such as the six nitrates, the four iodides, the two sulfocyanides, and acid sulfates. It is possible, however, to detect traces of regularity among the nitrates and sulfocyanides by putting into correspondence the phases which belong to the same crystalline system.

The variety of shapes possible for the individual curves is in striking contrast with the case for melting curves. Every rising melting curve


Phosphorus
AgNO3
NH4NO3
KNO3
RbNO3
CsNO3
TlNO3
NH4I
Cu2I2
AgI
HgI2
NH4HSO4
NH4SCN
KSCN
KHSO4
K2S
KClO3
KNO2
Pressure in Thousands of Kilograms per Square Centimeter.

is concave toward the pressure axis, and rises continuously, and the two known falling melting curves are also concave downward. We have here examples of rising and falling curves with curvature in either direction, curves with a maximum temperature (HgI_2), and curves with a maximum or minimum pressure (H_2O and Benzol). No cases have



been found of a critical point, however. In the diagrams several curves are indicated as coming to an end; this simply means that for one reason or another further measurements were impossible. The relatively high frequency of falling transition curves is in contrast to the case for melting. A falling curve means that the phase stable at the higher temperature has the smaller volume. There are only two known cases for melting, whereas more than one quarter of the cases above are of

this type. $KHSO_4$ affords a notable example where three curves of this type meet in a triple point. For a liquid, Δv always decreases with increasing temperature on either a rising or a falling curve. On the rising transition curves there are 37 cases of normal variation of Δv and 5 of abnormal variation; on the falling curves 8 normal and 8 abnormal cases.

The relative compressibility, thermal expansion, and specific heat of neighboring phases is significant. It is natural to expect that the phase of smaller volume will have the smaller compressibility and thermal expansion, and that the phase stable at the higher temperature will have the higher specific heat. If we call this behavior 'normal,' then on rising curves we find 9 cases of normal and 11 of abnormal compressibility, and on falling curves 1 normal and 7 abnormal. The expansion shows 5 normal and 7 abnormal cases on rising curves and 2 normal and 4 abnormal on falling curves. C_p is normal in 5 cases and abnormal in 7 cases on rising curves, and normal in 6 cases and abnormal in 1 on falling curves. The fact of abnormal C_p is of considerable significance from the point of view of the quantum hypothesis. It means (if we may apply the same considerations to C_p as to C_v, which is usually done) that the specific heat curves of the two modifications cannot be of the same character, but that somewhere between the transition point and absolute zero the one which is lower at the transition point must cross and lie above the other.

In addition to the substances enumerated above, about 100 others have been examined without finding other forms.

[1] P. W. Bridgman, *Proc. Amer. Acad.*, 47, 439-558 (1912); *Physic. Rev.*, 3, 126-203 (1914).

ON ISOTHERMALLY CONJUGATE NETS OF SPACE CURVES

By Gabriel M. Green

DEPARTMENT OF MATHEMATICS, HARVARD UNIVERSITY

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Bianchi[1] has called a parametric net of curves on a surface isothermally conjugate if, when the surface is referred to these curves, the second fundamental form, $D\,du^2 + 2D'\,du\,dv + D''\,dv^2$, may by a transformation $\bar{u} = U(u), \bar{v} = V(v)$ be made to take on the same shape as does the first fundamental form when the parametric net is isothermal; i.e., the parametric net is isothermally conjugate if $D' = 0$, $D = D''$. These nets have lately attained increased importance, so that Wilczynski's recent geometric interpretation[2] of Bianchi's condi-

tion is of great interest. In the present note, we propose to give a new and simple geometric characterization of isothermally conjugate nets which is entirely different from Wilczynski's.

Let $y^{(1)}, y^{(2)}, y^{(3)}, y^{(4)}$, be the homogeneous coordinates of a point in space, and let the four functions

$$y^{(k)}=f^{(k)}(u,v) \qquad (k=1,2,3,4) \tag{1}$$

define a surface S_y on which the curves u = const., v = const. form a conjugate net. Then the $y^{(k)}$'s satisfy a completely integrable system of two partial differential equations of the form[3]

$$\begin{aligned} y_{uu}&=a\,y_{vv}+b\,y_u+c\,y_v+d\,y,\\ y_{uv}&=b'y_u+c'y_v+d'y. \end{aligned} \tag{2}$$

The second of these is of the familiar Laplace type, characteristic of conjugate nets; the first shows that the conjugate net defined by equations (1) is isothermally conjugate if and only if

$$\frac{\partial^2}{\partial u\,\partial v}\log a=0. \tag{3}$$

The coefficients in equations (2) are not arbitrary, but are subjected to certain integrability conditions. One of the relations yielded by these conditions is that[4]

$$\frac{\partial}{\partial v}(b+2c')=\frac{\partial}{\partial u}\left(2b'-\frac{c}{a}-\frac{\partial}{\partial v}\log a\right),$$

or

$$b_v+2c'_v=2b'_u-\left(\frac{c}{a}\right)_u-\frac{\partial^2}{\partial u\,\partial v}\log a. \tag{4}$$

The minus first and first Laplace transforms of the point y are respectively

$$\rho=y_u-c'y, \qquad \sigma=y_v-b'y,$$

which represent covariant points on the tangents at y to the curves of the net passing through y. The surface S_ρ is the second focal sheet of the congruence of tangents to the curves v = const. on S_y, and S_σ is the second focal sheet of the congruence of tangents to the curves u = const. on S_y. Let us, with Wilczynski,[3] call the line $\rho\sigma$ corresponding to the point y the *ray* of the point y, and the totality of rays, which form a congruence, the *ray congruence*.

The osculating planes of the two curves u = const. and v = const. at a point y meet in a line which passes through y and which Wilczynski

calls the *axis* of the point y. The totality of axes, which correspond to all the points y of the surface S_y, constitute a congruence, the *axis congruence.*

We may write the first of equations (2) in the form

$$y_{uu} - by_u - dy = ay_{vv} + cy_v.$$

The left-hand member represents a point in the osculating plane to the curve v = const., and the right-hand member a point in the osculating plane to the curve u = const., at y. Therefore, since the coordinates are homogeneous, the point

$$z = y_{vv} + \frac{c}{a} y_v$$

lies on the line of intersection of the two osculating planes, and the line yz is the axis of the point y.

We may determine the developables of the axis congruence as follows. If the point y moves to the point $y + dy$, the point z moves to $z + dz$, where $dy = y_u\,du + y_v\,dv$ and $dz = z_u\,du + z_v\,dv$. We wish the line yz to generate a developable. This will happen if and only if the four points y, z, $y + dy$, $z + dz$ lie in a plane, or what is the same thing, if the points y, z, $y_u\,du + y_v\,dv$, $z_u\,du + z_v\,dv$ are coplanar. We have on differentiation of equations (2)

$$y_{uvv} = \alpha^{(12)} y_{vv} + \beta^{(12)} y_u + \gamma^{(12)} y_v + \delta^{(12)} y,$$
$$y_{vvv} = \alpha^{(03)} y_{vv} + \beta^{(03)} y_u + \gamma^{(03)} y_v + \delta^{(03)} y,$$

where in particular

$$\alpha^{(12)} = c', \quad \beta^{(12)} = b'^2 + b'_v, \quad \gamma^{(12)} = b'c' + c'_v + d,'$$
$$\alpha^{(03)} = b' - \frac{c}{a} - \frac{\partial}{\partial v} \log a, \quad \beta^{(03)} = \frac{1}{a}(b'c' + b'_u - b_v + d'), \tag{5}$$
$$\gamma^{(03)} = \frac{1}{a}[\,b'c + c'(c' - b) + c'_u - c_v - d\,],$$

so that on using these and equations (2) we find

$$z_u = y_{uvv} + \frac{c}{a} y_{uv} + \left(\frac{c}{a}\right)_u y_v$$
$$= c'y_{vv} + \left(\beta^{(12)} + \frac{b'c}{a}\right) y_u + \left[\gamma^{(12)} + \frac{cc'}{a} + \left(\frac{c}{a}\right)_u\right] y_v + (\,)y,$$
$$z_v = y_{vvv} + \frac{c}{a} y_{vv} + \left(\frac{c}{a}\right)_v y_v$$
$$= \left(b' - \frac{\partial}{\partial v} \log a\right) y_{vv} + \beta^{(03)} y_u + \left[\gamma^{(03)} + \left(\frac{c}{a}\right)_v\right] y_v + (\,)y,$$

in which the coefficients of y do not concern us. Consequently,

$$z_u du + z_v dv = \left[c' du + \left(b' - \frac{\partial}{\partial v}\log a\right) dv\right] y_{vv} + \left[\left(\beta^{(12)} + \frac{b'c}{a}\right) du + \beta^{(03)} dv\right] y_u + \left[\left(\gamma^{(12)} + \frac{cc'}{a} + \left(\frac{c}{a}\right)_u\right) du + \left(\gamma^{(03)} + \left(\frac{c}{a}\right)_v\right) dv\right] y_v + (\,)\, y.$$

Now, if the points y, z, dy, dz are to be coplanar, the determinant of the coefficients of y_{vv}, y_u, y_v in the expressions for z, dy, dz must vanish; on expansion this determinant yields the quadratic in $du : dv$,

$$a\left[\gamma^{(12)} + \left(\frac{c}{a}\right)_u\right] du^2 - \mathfrak{D}\, du\, dv - a\,\beta^{(03)} dv^2 = 0, \tag{6}$$

where, on using (4), we find

$$\mathfrak{D} = d + ab'^2 - c'^2 + b'c + bc' + ab'_v - c'_u. \tag{7}$$

The quadratic (6) determines the direction in which y must move, in order that the axis yz may trace out a developable; there are two such directions at each point of S_y. We may regard (6) as a differential equation defining a net of curves on S_y having the property that if the point y traces out a curve of this net, the corresponding axis generates a developable surface. We call the two curves of the net which pass through the point y the *axis curves* of the point y.

In like manner, we may determine the developables of the ray congruence, i.e., the net of curves on S_y having the property, that if the point y traces out a curve of the net, the corresponding ray traces out a developable of the ray congruence. The differential equation defining this net of curves, which we call the *ray curves*, is without difficulty found to be

$$a\,\mathrm{H}\, du^2 - \mathfrak{D}\, du\, dv - \mathrm{K}\, dv^2 = 0, \tag{8}$$

where $\mathfrak{D}$ is given by (7), and

$$\mathrm{H} = d' + b'c' - b'_u, \qquad \mathrm{K} = d' + b'c' - c'_v \tag{9}$$

are the Laplace-Darboux invariants of the given conjugate net.

If we use (9), we find from (5) that

$$a\,\beta^{(03)} = \mathrm{H} + 2b'_u - b_v, \qquad \gamma^{(12)} + \left(\frac{c}{a}\right)_u = \mathrm{K} + 2c'_v + \left(\frac{c}{a}\right)_u,$$

the latter of which becomes, on use of (4),

$$\gamma^{(12)} + \left(\frac{c}{a}\right)_u = \mathrm{K} + 2b'_u - b_v - \frac{\partial^2}{\partial u\,\partial v}\log a.$$

The differential equation (6) of the axis curves may therefore be written

$$a\left[\mathrm{K}+2b'_u-b_v-\frac{\partial^2}{\partial u\,\partial v}\log a\right]du^2-\mathfrak{D}\,du\,dv-(\mathrm{H}+2b'_u-b_v)\,dv^2=0. \quad (10)$$

The differential equation of the asymptotic curves is

$$a\,du^2+dv^2=0 \quad (11)$$

The pair of asymptotic tangents at y is of course harmonically separated by the tangents to the curves of our conjugate net. The differential equation

$$a\,du^2-dv^2=0 \quad (12)$$

defines a new net of curves. It evidently has the property, that the tangents to the two curves of the net at the point y separate harmonically both the pair of asymptotic tangents and the tangents to the two curves of our conjugate net. It is moreover the only net which has this property; since it also is a conjugate net, we call it the *associate conjugate net.*

We shall define another net of curves which will be of importance in our geometric interpretation. The quadratic

$$a\,\mathrm{H}\,du^2+\mathfrak{D}\,du\,dv-\mathrm{K}\,dv^2=0 \quad (13)$$

has for its roots the negatives of the roots of (8). It therefore defines a net such that the tangents to the two curves thereof at the point y are the harmonic conjugates of the two ray tangents (the tangents to the ray curves) with respect to the original conjugate tangents (the tangents to the curves of the original conjugate net). For convenience, let us call the curves defined by (13) the *anti-ray curves*, and the two tangents to the anti-ray curves at the point y the anti-ray tangents of the point y.

Let us now fix our attention upon a point y of the surface S_y, and let us regard equations (10), (12), and (13) as binary quadratics whose roots give respectively the pairs of axis tangents, associated conjugate tangents, and anti-ray tangents of the point y. The Jacobian of the forms (10) and (12) is

$$a\,\mathfrak{D}\,du^2+2a\left(\mathrm{H}-\mathrm{K}+\frac{\partial^2}{\partial u\,\partial v}\log a\right)du\,dv+\mathfrak{D}\,dv^2=0, \quad (14)$$

and its roots give the pair of lines through y which separate harmonically both the pair of axis tangents and the pair of associated conjugate tangents of y. The Jacobian of the forms (12) and (13) is

$$a\,\mathfrak{D}\,du^2 + 2a\,(\mathrm{H} - \mathrm{K})\,du\,dv + \mathfrak{D}\,dv^2 = 0 \qquad (15)$$

and defines the pair of lines through y which separate harmonically both the pair of anti-ray tangents and the pair of associate conjugate tangents of the point y.

The two Jacobians (14) and (15) coincide if and only if

$$\frac{\partial^2}{\partial u\,\partial v}\log a = 0, \qquad (3\,bis)$$

i.e., if and only if the original conjugate net is isothermally conjugate. We may state our result as follows:

A necessary and sufficient condition that a conjugate net of curves on a surface be isothermally conjugate is that at each point of the surface the pair of axis tangents, the pair of associate conjugate tangents, and the pair of anti-ray tangents be pairs of the same involution.

By means of the various nets of curves defined in the course of the above interpretation, we have been enabled to deduce a number of properties of isothermally conjugate nets. We have included this more extended discussion in a longer paper, which is a sequel to the one on conjugate nets to which reference has already been made.

[1] L. Bianchi, *Vorlesungen über Differentialgeometrie*, tr. M. Lukat, 2te Aufl., pp. 135 et seq.

[2] E. J. Wilczynski, *Trans. Amer. Math. Soc.*, **16**, 311–327 (1915).

[3] G. M. Green, *Amer. J. Math.*, **37**, 215–246 (1915). Cf. §1.

[4] Ibid., end of §3.

THE RÔLE OF THE LIVER IN ACUTE POLYCYTHAEMIA: THE MECHANISM CONTROLLING THE RED CORPUSCLE CONTENT OF THE BLOOD

By Paul D. Lamson

PHARMACOLOGICAL LABORATORY, JOHNS HOPKINS UNIVERSITY

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It is very generally considered by all except those who have paid special attention to the subject, that the number of red corpuscles per unit volume of blood is, in the normal individual, a fairly fixed quantity subject to gradual change only. A more careful study shows however that this number is subject to very rapid and great changes, and instead of being constant, that it is continually changing under physiological conditions.

Questions naturally arise as to what factors will cause a change in

the number of red cells;[1] where these changes take place; whether the increase in number is relative or absolute; and finally if there is a mechanism controlling the red corpuscle content of the blood, and if so what is its function.

It has been found that asphyxia in any form causes the number of red cells to increase. Reduced atmospheric pressure, reduced partial tension of oxygen, obstruction of the air passages, reduced oxygen capacity of the blood, as in carbon monoxide poisoning, or increased oxygen consumption as in exercise, will cause an increase in the number of red cells. Furthermore, obstruction to the circulation in the lungs, and consequent interference with the oxygenation of the blood, as in congenital heart conditions, will cause polycythaemia. I have been able to produce this form of polycythaemia experimentally by the injection of either corpuscles hardened with formaldehyde, or an inert powder as lycopodium, or oil, thus inducing embolism of the lungs and obtaining as great an increase in the number of red corpuscles as 3,000,000 in fifteen minutes.

Besides, it is known that various substances occuring physiologically in the body as epinephrin, pituitrin, carbon dioxide, and substances entirely foreign to the body as nicotine, radium chloride, etc., may produce very marked changes in the red count.

And finally I have been able to show that the number of red cells is under nervous control. In cats frightened by a dog for a minute or so, the red count rose one or two millions in five minutes or less, and from excitement alone reached the extreme value of 16,776,000 in one instance and 14,464,000 and 14,920,000 in two other cases.

There has been a great deal of work done concerning the magnitude of the changes in number of red cells and the haemoglobin content of the blood at high altitudes, and in exercise, but up to this time there has been no experimental evidence as to the seat of the changes which cause this increase. On this account the following experiments were systematically undertaken to find out if possible where the changes take place which cause an increase in the number of red cells.

For this purpose epinephrin was chosen as a means of producing polycythaemia, as it can be obtained in a pure state, accurately graduated in amount, and applied as a stimulus with certainty when intravenously injected. Furthermore it occurs normally in the body, and ether does not interfere with its action. When injected in doses of 0.9 mg. per kilo, intravenously, it produces with great regularity an increase of one or two millions in the red count in fifteen or twenty minutes.

To obtain uniform results the animals were etherized, and blood taken from either the jugular or femoral veins by means of a hypodermic needle, which method has been shown to give a true index of the red count in the generally circulating blood.

The entire intestine, its mesentery, the omentum, spleen and pancreas were removed singly, and in other cases altogether, after which epinephrin was injected. Epinephrin caused the same increase in number of red cells in these animals in which the above organs had been removed as in normal animals. It is apparent then that these organs are not the chief seat of the changes by which the red count is increased.

As the liver could not be removed except by the introduction of mechanical complications, its part in the production of polycythaemia was sought by other means.

In the first place animals were completely tied in two above the diaphragm. This is accomplished by opening the chest on both sides between two of the lower ribs, cutting the tissues to the sternum and back bone, and tying the great vessels, the sternum, back bone and adjoining tissues securely with strong ligatures. Except for what blood could pass up and down inside the vertebrae, the animal was completely tied in two above the diaphragm.

In such an animal, the injection of epinephrin caused no increase in the number of red cells.

An operation in which the animal was again tied in two, but this time placing the ligatures about the aorta just below the mesenteric artery and above the renal arteries, and completely tying the rest of the vessels and tissues at this level, gives an animal similar to the one above, except that the blood supply to the liver, spleen, intestine, mesentery, pancreas and omentum has been added. As we know that removal of all these organs except the liver, has no effect on the production of polycythaemia after the injection of epinephrin, we have for our purposes, in this case merely added the liver to the animal which was divided above the diaphragm.

Epinephrin injected into such an animal increases the number of red cells as in a normal animal.

From these experiments it appears that the liver is the seat of the changes which cause an increase in number of red cells. Further proof of this was obtained by quite different means.

On account of the irregularity with which the hepatic artery branches it was considered a more certain means of shutting off the arterial blood supply to the liver to ligate the coeliac axis artery, than to attempt ligation of the hepatic artery. The coeliac axis artery supplies the

liver, spleen and pancreas, and part of the stomach, but we have seen that removal of all of these except the liver has no effect on the production of polycythaemia after injection of epinephrin. If then the coeliac axis artery is ligated, and epinephrin injected, no change in the number of red cells is observed, but if this ligature is removed, even one-half hour after the injection of epinephrin, the red count will immediately increase, as if the usual dose of epinephrin had been injected into a normal animal.

A control experiment was done in which the coeliac axis artery was ligated, and after half an hour the ligature removed. This had no effect on the red count, which shows that mere interruption of the arterial circulation to the liver is not the only factor necessary to change the number of red cells.

I have also shown that ligation of the portal vein, as in the operation of removing the intestine, has no effect on the production of polycythaemia following the injection of epinephrin. Also in those cases in which the hepatic artery was ligated, epinephrin was carried to the liver by the uninterrupted portal circulation, and yet no change in the number of red cells took place.

Furthermore, in cases where the hepatic artery was tied, injection of epinephrin caused no increase in the number of red cells, although there was no interference with the blood supply to the kidneys, bone marrow, muscles, lungs or skin. These organs may then be considered as playing no part in the production of polycythaemia due to epinephrin injection.

It appears then that the liver is the organ capable of increasing the red corpuscle content of the blood after injection of epinephrin, and that the arterial blood supply to the liver must be intact to allow these changes to take place.

As to the changes which occur in the liver certain facts were observed in these experiments. There is a decrease in plasma volume, not however sufficient to account entirely for the increase in number of red cells. There are cells present in the circulation during polycythaemia which were not there before its production, as shown by a decrease in the average size of the red corpuscles, and a decrease in their percentage haemoglobin content. These cells show none of the usual signs of young cells. That is, they are not nucleated, they show no change in fragility, and they have no increased metabolism such as would result in an increased rate of reduction.

Concerning the mechanism which normally controls the red count, a few points of interest were observed.

It has been found that the red count is under nervous control, as shown by the increase in the number of red corpuscles after stimulation of the vago-sympathetic trunks, after asphyxia of the brain alone, and after emotional excitement as fear, rage, etc. It was also shown that the adrenals play a part in this mechanism, as epinephrin has been proved to be one of the most powerful means of increasing the number of red cells, and as the chief physiological stimuli which produce polycythaemia, namely asphyxia, exercise, fear, rage, etc., all cause a reflex stimulation of the adrenals, and an increased epinephrin output. Removal of the adrenals was found to exclude the production of polycythaemia in asphyxia, fear and rage, and after the injection of pituitrin, although these same animals responded to the injection of epinephrin by the usual increase in number of red cells. Furthermore, removal of the adrenals is followed after several hours by a complete loss of control of the red count.

When the number of red cells is increased in acute polycythaemia, we have seen that there is also an increase in haemoglobin, but not in proportion to the increase in number of red cells. This being the case, the blood is better equipped not only to carry more oxygen, but on account of the increased surface of the haemoglobin, to take up more oxygen per unit of time. The body is then better prepared to maintain a constant oxygen content of the blood under various physiological and pathological conditions.

We may therefore conclude that there is in the body a mechanism for regulating the red corpuscle content of the blood; that this mechanism is under nervous control, responding to nervous, chemical and emotional stimuli; that the adrenal glands play a part in this mechanism, and that the liver is the seat of the changes which increase the number of red cells, partly by a reduction in plasma volume, and partly by bringing cells into the circulation which are not normally present.[2]

[1] The terms red count, number of red cells, etc., will be used for the sake of brevity instead of the more exact phrase "Number of red corpuscles per unit volume of blood."

[2] This article is given in full in the *Journal of Pharmacology and Experimental Therapeutics*, Vol. 7, Nos. 1 and 2 (1915).

THE POTENTIALS AT THE JUNCTIONS OF SALT SOLUTIONS

By Duncan A. MacInnes

LABORATORY OF PHYSICAL CHEMISTRY, UNIVERSITY OF ILLINOIS

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A concentration-cell consisting of two identical reversible electrodes in contact with two differently concentrated solutions of the same salt gives an electromotive force which is the algebraic sum of three potentials—those at the two electrodes and that at the junction of the solutions. Since for most theoretical purposes the potential at the electrodes alone is desired, many attempts have been made to evaluate the potential at the liquid junction. The effect of interposing strong salt solutions on the electromotive force of the cell has also been studied with the view of eliminating the effect of the liquid junction.

It has been usual to test the correctness of the value assigned to the potential at the junction, or the efficacy of the artifice employed in eliminating it, by seeing whether the resulting electromotive force E at the electrodes can be calculated by the equation $E = (RT/F)\ln(C_1/C_2)$ where C_1 and C_2 are the concentrations of the ions in the two solutions as calculated from conductance measurements, and R, T, and F are the gas-constant, the absolute temperature, and the faraday respectively. Recent work[1] has indicated, however, that the voltages calculated by this formula are too high. It therefore seems desirable to investigate the subject of the liquid junction connecting two solutions of the same salt with a view of finding an expression, involving the fewest possible assumptions, for its electromotice force.

If the cell Ag, AgCl (solid) + KCl (0.1n), AgCl (solid) + KCl (0.01n), Ag operates reversibly until one faraday passes through it, one equivalent of chloride-ion enters the dilute solution and the same amount of chloride-ion is electrolyzed out of the more concentrated solution. The current is carried across the liquid junction by the movement of n_c equivalents of potassium-ion in the direction of the current and by the migration of $(1 - n_c)$ equivalents of chloride-ion in the reverse direction, n_c being the transference-number of the cation. The total effect of the passage of the faraday of electricity is the transference of n_c equivalents of salt, or $2n_c$ equivalents of ion, from the 0.1 normal KCl solution to the 0.01 normal KCl solution. The accompanying electrical work is the product $E\,F$ where E is the total E. M. F. of the cell and F is the value of the faraday. Now the electrical work and consequently the electromotive force at the liquid junction will be determined by the

algebraic sum of the number of equivalents of ion that are carried across it from the concentrated to the dilute solution, in this case equal to $n_c - (1 - n_c) = 2n_c - 1$. If we now make the assumption that the osmotic work involved in the transferring of a gram equivalent of positive ion from a dilute to a concentrated solution is the same as the work necessary for the transfer of a corresponding amount of negative ion, we can obtain the electromotive force at the liquid junction by the simple proportion:

$$(E_L F : EF = (2n_c - 1) : 2n_c \text{ or } E_L = E\,(1 - 1/2n_c). \qquad (1$$

This equation contains only the directly measurable quantities E and n_c, and contains no assumption concerning the concentrations of the ions in the two solutions.

A direct test of this simple equation is not possible; but an indirect one is afforded by the following considerations. The electrode-potentials of cells of the type Ag, AgCl (solid) + MCl (C_1), AgCl (solid) +MCl (C_2), Ag would be expected to be the same whether hydrogen or any one of the alkali metals is chosen for the radical M, if the concentrations of C_1 and C_2 are the same in each case and below about 0.05 normal. The sum of the electrode-potentials is determined by the difference of the osmotic pressures of the chloride ions in the two solutions; and this difference of osmotic pressure is, probably, nearly the same for dilute solutions of chlorides of univalent cations at corresponding concentrations, since the degrees of dissociation in dilute solution as determined by the conductivity method have been found to be nearly the same for these substances. If this is true, and if the assumption involved in the above-given expression for the potential at the liquid junction is correct, the sum of the electrode-potentials should be independent of the nature of the cation. This amounts to the assumption that the free energy of dilution of the chloride ion is the same whether hydrogen, potassium, or sodium is the cation, since the process at the electrodes during the operation of a cell consists in the formation of one equivalent of chloride ion in the dilute solution and the removal of the same amount of chloride ion from the concentrated solution per faraday passed through the cell. Jahn's accurate work (loc. cit.) on concentration cells with hydrochloric acid, potassium chloride, and sodium chloride is, fortunately, well adapted to test these conclusions.

Table I, which is self explanatory, gives the result of my calculations, based on Jahn's electromotive-force data, which are here given in millivolts. The transference-numbers are from Noyes and Falk's[2] compilation.

TABLE I

CALCULATION OF THE LIQUID JUNCTION POTENTIAL AND ELECTRODE POTENTIALS (IN MILLIVOLTS) OF CHLORIDE CONCENTRATION CELLS WITH SILVER—SILVER CHLORIDE ELECTRODES

SUBSTANCE	MOLS PER LITER	TRANSFERENCE NUMBER	E. M. F. OF CELL E	LIQUID JUNCTION POTENTIAL, E_L	ELECTRODE POTENTIAL $(E - E_L)$	AVERAGE DEVIATION FROM MEAN
HCl.....	0.01665 0.001665	0.833	−92.35	−36.94	−55.41	
NaCl.....	0.01673 0.001674	0.396	−43.60	+11.46	−55.06	0.24
KCl.....	0.01670 0.001674	0.496	−54.24	+00.44	−54.69	
HCl.....	0.03330 0.003329	0.833	−91.62	−36.64	−54.98	
KCl.....	0.03347 0.003347	0.496	−54.03	+0.43	−54.47	0.25
HCl.....	0.008315 0.001665	0.833	−64.87	−25.95	−38.92	
NaCl.....	0.008364 0.001674	0.396	−30.73	+ 8.08	−38.81	0.04
KCl.....	0.008329 0.001670	0.495	−38.44	+ 0.39	−38.83	
HCl.....	0.006686 0.001665	0.833	−56.14	−22.45	−33.79	
NaCl.....	0.006686 0.001674	0.396	−26.53	+6.97	−33.49	0.08
KCl.....	0.006700 0.001670	0.495	−33.30	+0.34	−33.64	

It will be observed that in each group of cells in which the solutions of electrolytes have the same concentrations, the calculated sum of the electrode-potentials has the same value within a few tenths of a millivolt. This is true even though the liquid-junction potential in the case of the hydrochloric acid cells is 40% of the total electromotive force, and of opposite sign to that in the case of the sodium chloride solutions. The agreement is as close as can be expected from our knowledge of the transference-numbers. Calculations based on other work by the same author show a similar agreement.

This result indicates, at any rate in the case of solutions of concentrations up to 0.03 normal, the correctness of the two assumptions involved in the calculations, namely, that the osmotic work of transferring equivalent quantities of the ions is nearly the same for positive ion and negative ions, and that the osmotic work of transferring chloride-ion from one salt concentration to another is nearly the same whether the cation is potassium, sodium, or hydrogen. It is probable that the first of these assumptions is substantially correct up to much higher concentrations than it can be tested by the method here employed, since this method involves also the second assumption just stated, which doubtless becomes inexact at higher concentrations. Equation (1) here considered must in any case give a much more exact value of the liquid potential than does the Nernst equation

$$E_L = \frac{RT\ (1-2n_c)}{F} \ln \frac{C_1}{C_2}.$$

How much the values calculated by the latter differ from those calculated by the former equation in the case of the more concentrated of these dilute solutions is shown in the following table:

SUBSTANCE	MOLS PER LITER	LIQUID POTENTIAL IN MILLIVOLTS		DIFFERENCE
		By equation (1)	By Nernst equation	
HCl	0.01665			
	0.001665	−36.94	−38.03	1.09
NaCl	0.01673			
	0.001673	11.46	11.72	0.26
KCl	0.01670			
	0.001674	00.44	00.45	0.01
HCl	0.03330			
	0.003329	−36.64	−38.23	1.59
KCl	0.03347			
	0.003347	0.43	0.45	0.02

The difference between the values for the liquid junction potential as calculated by the two methods is small for salts having ions with nearly the same mobility, but the difference increases rapidly with increased concentrations of the solutions, since the Nernst equation contains the assumption that the ions are 'perfect' solutes, an assumption which is not even approximately true at higher concentrations if the ion con-

centrations are calculated in the usual manner from conductance measurements.

In conclusion it may be noted that the value of E/n_e in equation (1) when multiplied by F, the value of the faraday, represents the maximum work or free-energy change attending the transfer of one equivalent of salt from one concentration to the other, and that therefore E_L may be calculated from any of the properties related to osmotic pressure, such as freezing-point. This free energy-change may also be obtained directly from electromotive force measurements of cells without transference of the type;Ag, AgCl (solid) + KCl (0.1n), $K(Hg)_x$ − $K(Hg)_x$, AgCl (solid) + KCl (0.01n), Ag, investigated by MacInnes and Parker.

[1] See for instance, Jahn, *Zs. physik. Chem.* 33, 545 (1900); Tolman and Ferguson, *J. Amer. Chem. Soc.* 34, 232 (1912); MacInnes and Parker, *J. Amer. Chem. Soc.* 37, 1445 (1915).

[2] *J. Amer. Chem. Soc.* 33, 1454 (1911).

A STATISTICAL STUDY OF THE VISUAL DOUBLE STARS IN THE NORTHERN SKY

By Robert G. Aitken

LICK OBSERVATORY, UNIVERSITY OF CALIFORNIA

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The double star survey, initiated in April, 1899,[1] has now been completed to −14° Declination for the winter sky and to −22° Declination for the summer sky. This is about as far south as atmospheric conditions make it desirable to extend the survey at Mount Hamilton.

The original plan contemplated the examination with one of the refracting telescopes of the Lick Observatory of all stars as bright as 9.0 magnitude in the *Bonn Durchmusterung*, the identification of all previously known double stars, and the cataloguing of any new pairs, not exceeding 5″.00 in angular separation. Limits must be imposed in work of this character, and those chosen, while necessarily arbitrary, were selected after careful study as being liberal enough for the purpose in mind, which was the accumulation of data for a statistical study of the number and distribution of the visual double stars.

The survey has yielded 4300 new double stars, 1328 discovered by Prof. W. J. Hussey, the others by the writer. Twenty-eight of these have angular separation slightly exceeding 5″.00; 3199, or 74.4% of the whole number, fall within the limit 2″.00, and 1280 (29.8%), within the limit 0″.50. The distance limit adopted has thus been carefully observed, but for various reasons many stars fainter than the limit 9.0 were ex-

amined with the result that several hundred of our discoveries are fainter than this magnitude. It may develop that the most important contribution made by the survey, possibly the only one of permanent value, is this great addition to the number of known close visual binaries. Nevertheless, the statistical study has been the object which I have held definitely in mind from the beginning and I have regarded discovery as incidental.

The present paper gives the results from a partial statistical study for the northern half of the sky, the data including all visual double stars (as distinguished from spectroscopic binaries) as bright as 9.0 B. D. magnitude which fall within the limits set by my 'working definition' of a double star.[2] In all there are 5400 such pairs north of the celestial equator, 2823 of which were discovered in the course of the Lick Observatory survey. It is not claimed that this number is exhaustive, but it may fairly be said that it represents the capacity of the combination of telescope and observer under *average good observing conditions at Mount Hamilton.* Unfortunately the data are not strictly homogeneous, for part of the survey was carried out with the 12-inch refractor. A study of the relative efficiency of the 12-inch and 36-inch refractors in double star discovery leads to the conclusion that, if the entire work had been done with the latter telescope, about 250 additional pairs would have been discovered in the northern hemisphere.

According to Seeliger's count, the *Bonn Durchmusterung* contains 100,979 stars as bright as 9.0 magnitude north of the celestial equator; 5400 of these, or one in 18.7 on the average, have actually been found to be double within the chosen limits. If we add only 200 more pairs the ratio is increased to 1 in 18.03. We may therefore conclude that *at least one in every eighteen, on the average, of the stars as bright as 9.0 magnitude in the northern half of the sky is a double star visible with the 36-inch telescope.*

Table I shows the distribution of these double stars in galactic latitude by magnitude classes, the arguments, for convenience of comparison, being those adopted by Seeliger[3] in his discussion of the B. D. stars. The zones are 20 wide, the first extending from the north galactic pole to $+70°$, the fifth from $+10$ to -10 galactic latitude. Zone IX, touching the south galactic pole, lies entirely south of the celestial equator. The progressive increase in numbers in every magnitude class as we approach Zone V from the north or from the south was to be expected, for it is well known that the stars as a whole show a similar distribution. When, however, we compare the results with Seeliger's tabulation for all the B. D. stars to 9.0, we find that the frequency curves

of double stars of every magnitude class rise to sharper maxima in the Milky Way than do the corresponding curves of the stars in general.

This is most strikingly exhibited in Table II which gives the percentage of double stars in the Milky Way zone, the 20° zone on either side of it, the area north of +30° and that south of −30° galactic latitude.

TABLE I

THE DISTRIBUTION OF DOUBLE STARS BY MAGNITUDE CLASSES AND ZONES OF GALACTIC LATITUDE

ZONE	MAG. TO 6.5	MAG. 6.6–7.0	MAG. 7.1–7.5	MAG. 7.6–8.0	MAG. 8.1–8.5	MAG. 8.6–9.0	TOTAL
I.....	19	13	14	29	40	84	199
II.....	43	28	50	68	114	193	496
III.....	60	43	56	79	148	254	640
IV.....	96	54	81	132	232	401	996
V.....	121	88	133	249	376	653	1620
VI.....	84	51	81	134	221	395	966
VII.....	28	23	18	54	90	154	367
VIII.....	7	6	5	12	31	55	116
Total.....	458	306	438	757	1252	2189	5400

Areas of all galactic latitudes were examined in winter, others are as in summer; the larger part of the work done with the 12-inch telescope lies between +27° and −3° galactic latitude; nearly the entire area south of −3°, and also the high northern galactic latitudes, were surveyed with the 36-inch refractor. The apparent increase in percentage in the Milky Way zone is therefore not due to the use of different telescopes nor to any other peculiarity in the collection of the data, and we must conclude that *close visual double stars are relatively more numerous in the Milky Way than elsewhere in the sky.*

TABLE II

PERCENTAGES OF DOUBLE STARS

GALACTIC LATITUDE	B. D. STARS TO 9.0	DOUBLE STARS	PERCENTAGE OF DOUBLE STARS
+90° to +30°	26948	1335	4.95
+30 to +10	19355	996	5.15
+10 to −10	26477	1620	6.13
−10 to −30	17831	966	5.13
−30 to −70	10368	483	4.66

I have tabulated the double stars in distance classes by steps of 0″.5 and find that the increased density toward the Milky Way is exhibited by stars of every class, though the percentages in Table II are most strongly affected by the close pairs because of their greater number.

Table III shows the relation between magnitude and angular separation. Since the progressive increase in the number of double stars as the angular distance diminishes is shown by stars of every magnitude class, it cannot be explained as merely a perspective effect, the more distant pairs appearing at smaller angles, for according to our present conceptions, stars of a given magnitude (e.g., from 8.6 to 9.0) are approximately at the same average distance from us. The relative angular separations of the double stars of this magnitude therefore reflect the relative linear distances between the components and it follows that *visual double stars as a rule revolve in relatively small orbits.*

The spectral classification of the majority of the 5400 stars under discussion is at present unknown. I have compared the list with the Draper Catalogue in volume 50 of the *Harvard Annals*, thus securing

TABLE III

THE DISTRIBUTION OF DOUBLE STARS IN THE NORTHERN HEMISPHERE BY ANGULAR DISTANCE AND MAGNITUDE

DIST.	0".00 TO 0.50	0".51 TO 1.00	(0".00) TO (1.00)	1".01 TO 2.00	2".01 TO 3.00	3".01 TO 4.00	4.01 TO 5.00	5".01 AND OVER
Mag. to 6.5	75	63	(138)	83	62	41	31	99
6.6 to 7.0	82	52	(134)	59	42	40	21	14
7.1 to 7.5	103	67	(170)	99	64	48	31	29
7.6 to 8.0	178	132	(310)	164	107	85	63	26
8.1 to 8.5	310	223	(533)	285	173	128	111	21
8.6 to 9.0	508	413	(931)	532	317	217	191	11
Total.....	1256	954	(2206)	1222	765	559	448	200

the spectral data for the brighter stars, and Prof. E. C. Pickering and Miss Annie J. Cannon have been kind enough to compare the list of double stars north of $+60°$ and that of the double stars in the first two hours of Right Ascension with the new Draper Catalogue now in course of preparation at Harvard, which is designed to extend to 9.0 magnitude. Combining the data from these three sources and excluding duplicates we have Table IV.

The distribution of these stars with respect to the galactic plane closely resembles that of the stars in general; Classes B and A crowd toward that plane, classes F, G and K show a more uniform distribution. The totals show how marked is the preference of the visual double stars for Classes A and F. It may be added that 46 of the 83 Class B stars belong to the subclasses B8 and B9 and that 104 of the 124 Class K stars are marked 'K' or 'K0.' Clearly, *close visual double stars are rare among stars of either very early or very late spectral class.* This confirms my con-

clusion, in 1910,[4] based on an analysis of the spectral classification of 164 of the more rapidly moving binary stars on my observing program.

The ten stars of Classes M and Oe5 are obviously exceptional, and the number is too small for any discussion of value. The following points however may be noted: The stars are all bright; the proper motions are very small; orbital motion has been observed in only one of them (η *Geminorum*), and in this system it is very slow; the companion in each instance is much fainter than its primary, and the angular separation between the components is neither very small nor very large. Probably they are all very distant.

TABLE IV

SPECTRAL CLASSIFICATION OF DOUBLE STARS

ZONE	SPECTRAL CLASS						
	B	A	F	G	K	M	Oe 5
I.....	0	8	4	2	3		
II.....	2	19	14	5	12		
III.....	3	37	59	28	22		
IV.....	18	81	68	45	26	1	
V.....	47	101	54	31	24	5	2
VI.....	9	84	47	31	15	1	1
VII.....	3	19	33	16	14		
VIII.....	1	8	29	16	8		
Total.....	83	357	308	174	124	7	3

The percentage of very close double stars—under 0″.50—is considerably greater in Classes B, A and F than in Classes G and K, but the percentage of pairs over 2″.0 is about the same in all classes except K, in which it is larger. To discuss the correlation between angular separation and spectral class we need further data. I hope to take up this question and others in a later paper.

The conclusions here presented of course apply only to the data upon which they rest, and, in view of the many warnings we have had against basing 'laws of nature' upon incomplete statistics, I refrain from any comment upon the bearing they may have upon current theories of the evolution of double star systems. It is my opinion that we have not yet sufficient knowledge of the facts to establish any theory with reasonable certainty.

[1] "On Double Stars," R. G. Aitken, *San Francisco, Pub. Astr. Soc. Pac.*, **16**, 235 (1904).

[2] "The Definition of the Term Double Star," R. G. Aitken, *Ast. Nachr.*, **188**, 281 (1911).

[3] "Ueber die Vertheilung der Sterne auf der nördlichen Halb-kugel . . . ," H. Seeliger, *München, Sitz. Ber. Ak. Wiss.*, 1884, Heft 4.

[4] "Second Catalogue of Spectroscopic Binary Stars," W. W. Campbell, *Lick Obs. Bull.*, **6**, 41 (1910).

WALNUT MUTANT INVESTIGATIONS

By Ernest B. Babcock

DEPARTMENT OF AGRICULTURE, UNIVERSITY OF CALIFORNIA

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About fifteen years ago there appeared in a nursery of Southern California Black Walnut seedlings a new and very distinct form of *Juglans*. Two of the original trees still stand where first transplanted from the seed bed. These are the type specimens which I described[1] and named *Juglans californica* var. *quercina* because of their general resemblance to a small-leaved oak. Similar seedlings have been secured from two other trees of *Juglans californica* Wats. In 1914 seedlings of this same oak-like type appeared in a nursery of the Northern California Black Walnut, *J. californica* var. *hindsii* Jepson. This is a very interesting parallel mutation because *hindsii* was originally described as a species[2] and is separated from *californica* by several distinct features. However, this occurrence of parallel mutations does not in itself necessitate any change in nomenclature other than to designate *quercina* as mutation rather than variety. Therefore I propose to designate *quercina* individuals produced from seeds of *J. californica* as *J. californica* mut. *quercina* and to similar individuals produced from *hindsii* seeds as *J. californica* var. *hindsii* mut. *quercina*.

In 1910 I discovered that many otherwise normal trees of Southern California Black Walnuts produce a second crop of flowers, from one to two months later than the first crop and that these nuts are invariably teratological.[3] However, up to 1913 all the plants produced by the teratological nuts collected resembled *J. californica*, i.e., there was no evidence that teratology was the cause of the origin of *quercina*.

In 1912 I was able to locate an apparently normal tree of *Juglans californica* Wats., which produces a small percentage of *quercina* seedlings each year and to learn that these grew from normal nuts. This tree is designated as Garden Grove No. 16. It is probably the only perennial mutating individual accessible for experimentation. That the mutation always occurs in the pistillate flowers is shown by the fact that Garden Grove No. 16 is the only tree in a row of 21 (all of which were tested in 1912) that produced *quercina* seedlings. An objection has been raised to the designation of Garden Grove No. 16 as a mutating individual. The argument advanced is based on the assumption that all new types of organisms are due to recombinations of genetic factors and hence that if Garden Grove No. 16 produces two types of seedlings

it must be a hybrid. To this objection I can only reply that thus far I have been unable to discover any morphological evidence that Garden Grove No. 16 is a hybrid.

In 1913 I gathered about 350 clusters of nuts from this tree and grew each lot separately. Among them were several clusters of teratological nuts. A single *quercina* seedling appeared in each of 42 of the normal clusters while three of the teratological clusters produced two or more *quercina* seedlings in each cluster. These results indicate that, while teratology *per se* is not the cause of the mutation, yet in a tree capable of producing *quercina* seedlings the mutation occurs more frequently among teratological than among normal flowers. The fact that in all the normal clusters producing *quercina* seedlings only one mutant came from each cluster, at once raised the question as to whether a certain location of the flower in the pistillate catkin might be associated with the occurrence of the mutation. This question is not yet fully answered but it is known that at least two different locations in the catkin are associated with the mutation and hence it can be stated definitely that no certain location of the pistillate flower is exclusively associated with the occurrence of the mutation.

On account of the interesting differences in chromosome numbers found among some of the evening primroses, we have determined the number in the somatic cells of both types of seedlings produced by Garden Grove No. 16. By planting one seed in a small pot excellent root-tips for cytological study are thrust out through the hole in the pot. The chromosomes are very small and cells that show satisfactory figures for counting chromosomes are comparatively rare. However, I am satisfied that the number of chromosomes in both types of seedlings is thirty-four. If further study reveals the same number of chromosomes in both *quercina* and *californica* seedlings secured from other sources, we must conclude that the mutation producing *quercina* is not due to a change in the number of chromosomes. The chromosome numbers for *hindsii* and its *quercina* mutants have not yet been determined.

Meanwhile, efforts have been made to test the genetic relation of *quercina* to *californica* by crossing. I have eight F_1 trees of the cross *J. californica* ♀ × *J. californica* mut. *quercina* ♂, all of which resemble *J. californica*. They produced some open pollinated nuts in 1914 and among the progeny of one of these eight (08 H_1F_1P8c) there has appeared one *quercina* seedling. This indicates that at least one of the eight is a veritable hybrid and not a parthenogen as might be the case judging from the results of certain crosses with oak pollen also made in 1908. Hence it is possible that the ratios produced in future tests of this indi-

vidual will throw some light on the nature of the mutation which produces *quercina*. Efforts are being made to secure self-pollinated nuts from each of these trees and in 1916 I hope to secure sesqui-hybrids from the cross *J. californica* mut. *quercina* ♀ × 08 H_1F_1P8c ♂. The completion of these breeding tests is especially desirable in view of the diverse behavior of certain of the original *quercina* trees. One of the type individuals breeds true, while a cotype individual produces both *quercina* and *californica* seedlings and another cotype individual produces nothing but *californica* seedlings.

The facts reported in this paper may be interpreted in part as meaning (1) that the mutation takes place in female flowers only and appears in the first generation after the mutation occurs but upon crossing with the species type it is completely recessive in the F_1 generation; (2) that the nature of the mutation is such that only certain genetic factors are affected without having the chromosome number disturbed.

Another walnut mutant has appeared in the form of a laciniate-leaved type of *Juglans regia*. Open pollinated nuts occasionally reproduce the new type. It is expected that self- and cross-pollinations will be made in 1916 and possibly cytological investigations.

[1] Babcock, E. B., Studies in Juglans I. Study of a New Form of *Juglans californica* Wats., *Univ. Cal. Pub. Agr. Sci.*, **2**, no. 1 (1913).

[2] Jepson, W. L., in *Bull. So. Cal. Acad. Sci.*, **7**, 23 (1908).

[3] Babcock, E. B., Studies in Juglans II. Further observations on a New Variety of *Juglans californica* Wats., and on Certain Supposed Walnut-Oak Hybrids, *Univ. Cal. Pub. Agr. Sci.*, **2**, no. 2 (1914).

HEREDITARY FRAGILITY OF BONE

By C. B. Davenport and H. S. Conard

CARNEGIE INSTITUTION OF WASHINGTON AND GRINNELL COLLEGE, IOWA

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While a weakness or brittleness of the long bones may arise from a number of pathological conditions affecting the bones, yet there has been recognized for 80 years a constitutional tendency to brittle bone that runs through families in such fashion as to demonstrate the presence of an hereditary factor. To it has been given the name fragilitas ossium or osteopsathyrosis.

In a classical case of osteopsathyrosis the femur is frequently found fractured at birth, but it mends quickly and smoothly. Repeatedly in life a slight knock causes a bone of the leg or arm to break; and sometimes before puberty the individual has suffered a score or more of

fractures. The tendency is often outgrown after puberty. Lovett and Nichols[1] have shown that in osteopsathyrosis the periosteal bone formation especially is imperfect, since the Haversian systems are not formed.

An examination of a large number of family histories shows clearly that heredity in osteopsathyrosis is typically direct—i.e., the factor that determines the irregular bone formation is a dominant one. The view that it is due to "some disturbance of metabolism of the mother" is disproved by the instance of twins, described by Schwarz and Bass,[2] one of whom showed osteopsathyrosis at birth while the other did not. The rare exceptions to the rule that a generation is not skipped are explained by the well known principle of imperfection of dominance or possibly to a lack of knowledge of an infantile condition of a parent.

An association of osteopsathyrosis with blue sclerotics has often been pointed out. The condition of blue sclerotics also appears to be a dominant trait. It is barely possible that the association is not a necessary one but on the other hand, there may be a linkage between these traits; pedigrees are not yet extensive and numerous enough to decide.

That osteopsathyrosis depends upon a single dominant factor is further indicated by the fact that the proportion of children in an affected fraternity that shows osteopsathyrosis is approximately half—actually 55%.

The presence of biotypes of osteopsathyrosis can be recognized. In some families the slightest pressure results in fracture; in other families the bones are fairly resistant. The commonest bone to break is the femur. But in some families the humerus seems especially weak and in still others the clavicle is frequently broken. Thus, associated with the imperfect bone development are special family traits that modify the result.

It appears, to summarize, that of a parent who was in early life osteopsathyrotic at least half of the children will be similarly affected. But if neither parent, though of affected stock, has shown the tendency then expectation is that none of the children will have brittle bones. Moreover, when a parent is affected it is possible that his children will show the tendency at about the same time of life, in the same bones and to the same degree as he himself showed it.

The full paper with numerous pedigree charts will appear in the Bulletin of the Eugenics Record Office.

[1] Lovett, R. W. and Nichols, E. H., *Brit. Med. J., London*, 1906, II, 915.
[2] Schwarz and Bass, *Medical Record*, 1912, p. 317.